ON

THE PATHOLOGY

OF

CHOLERA COLLAPSE.

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To what, at its onset, is due the collapse stage of cholera? Should this question ever be satisfactorily settled, we may still be as far off as ever from diminishing the fearful death-rate of the disease. Nevertheless, most authors who have written on cholera have endeavoured to frame some theory on the subject, more or less supported by facts, either for the simple purpose of arriving at a closer appreciation of the malady, to suggest a line of treatment to be pursued, or to justify that which had already been adopted. Although an erroneous theory does not necessarily lead to wrong practice, it is very likely to do so. It is from this belief that I have endeavoured, in the following pages, to indicate what to my mind appears to have claims to be considered the efficient cause of the Algide stage of cholera.

Even in the most rapidly fatal cases of the disease the stage of collapse is not that which first calls for observation. However rapidly it may supervene, there are either symptoms before death, or post-mortem evidences to show that it is not the primary result of the cholera poison. Now, although the first evidences of the attack may be most conveniently expressed as caused by some poison, the nature of which it is not my intention here to discuss, still remains to be proved whether, among the primary effects of the poison, conditions are not called into existence in the body upon which mainly depends the development of the algide stage of cholera, rather than upon the continuance of any peculiar action of the original specific cause or poison; whether, in fact, secondary causes do not come into operation, of a more familiar character, and more readily to be dealt with.

It is very generally admitted by some of the most distinguished writers on cholera that the symptoms of the so-called secondary fever are more the result of the previous profound derangement of the system than of the presumed continued action of a cholera poison. I am of opinion that this view may be carried much further, and that in endeavouring to express the relation of the various stages of cholera we must keep fully in view the doctrine of primary and secondary causes. Just as in explaining the revolutions of the heavenly bodies, one cause—viz., projectile impulse—is considered to have acted once and for all, after which their course is determined by secondary causes expressed by laws affecting all matter in common, so I believe, in cholera, having once granted a certain action of a special cause—the cholera poison,—we must endeavour to explain the subsequent phenomena of the disease in accordance with the known laws of the effects of certain diseased actions on the

body, without having continually to speak of this or that phenomenon as produced by an original specific cause.

Keeping the above considerations in view, I will proceed to review those effects which intervene between the commencement of the disease and the development of the algide state, and to see which of them may be deemed worthy of being regarded as an efficient cause of collapse.

First, As regards the drain of fluid from the blood. In the majority of cases of cholera the profuse alvine discharges of rice-water fluid form so prominent a symptom, that early in the history of the pathology of cholera they are found to have been regarded as the main cause of collapse. Subsequently this view was abandoned as at variance with those cases, though rare ones, in which the drain from the bowel was very inconsiderable, though they were attended with profound and rapidly fatal collapse. The value of the "Notes on Cholera," lately published by Dr George Johnson, greatly depends on the prominence he has given to such cases, which are very essential to be remembered in discussing the causation of cholera collapse. When an epidemic such as the present is setting many minds new to the inquiry to attempt the solution of the problem, Dr Johnson's little work comes very opportunely, to prevent time being wasted through the attention being directed into the wrong channel. He cites part of the following quotation from p. 130 of the "Report of the College of Physicians on the Morbid Anatomy and Pathology of Cholera, 1853." "Cholera poison is not known to produce its fatal effects without the characteristic affection of the intestines. Cholera sicca, in a strict sense, does not occur, for although the disease may be fatal without any evacuation the intestines after death, in such cases, have been found to contain the rice-water fluid. In one instance of this kind which came under our observation, on a post-mortem examination, the large intestine contained healthy fæces, whilst at the upper two-third of the small intestine the mucous membrane presented the ordinary changes induced by the cholera process, and the rice-water effusion was abundant."

One similar case has come under my own notice, namely, that of a boy, admitted into the London Fever Hospital, July 30, in profound cholera collapse. His father had died of the same disease the day before. No distinct history was obtainable, but the boy had vomited freely on his way to the hospital. He died two minutes after admission. A post-mortem examination showed the rectum to be occupied by nearly formed fæces, while the small intestines were filled with a considerable amount of grumous rice-water material, mucus, and some pale pink, strawberry-ercoloured fluid. The small intestines themselves were intensely congested, pink, fleshy, and œdematous. From these and numerous nearly similar cases, mentioned in the "Report" above quoted, which show that there is no relation between the amount of alvine discharges and the rapidity of development or profoundness of the collapse, it may now, I think, be taken as a set

point, that although the profuse discharges must necessarily have considerable influence on the course of the disease, they cannot be regarded as the efficient cause of cholera collapse.

The theory of the algide state being due to the contraction of the pulmonary artery and its branches, next calls for consideration. The prominent position recently given to this view is due to its enunciation by Dr Johnson in the work mentioned above. Having performed a real service in giving a *coup de grace* to the already nearly abandoned purgation theory of the causation of collapse, he has proceeded to frame an explanation of the onset of the algide symptoms on the basis of two assumptions. 1*st*, That there is a definite cholera poison circulating in the blood; and, 2*dly*, That it is the special function of that poison, through the blood, to cause contraction of the pulmonary artery and its branches, whereby the amount of blood sent to the lungs is diminished, to the effects of which he refers the collapse stage. There is no mistaking the rigorous absoluteness with which Dr Johnson states these views. At page 49, he says, "The blood thus poisoned excites contraction of the muscular walls of the minute pulmonary arteries, the effect of which is to diminish, and in fatal cases to entirely arrest, the flow of blood to the lungs." Again, at page 55, "The most interesting and conclusive evidence that arrest of blood in the lungs is the true key to the pathology of choleraic collapse is to be found in the simple yet complete explanation it affords of all the most striking chemical phenomena of the disease." I will quote one more passage from p. 68: "The remarkable arrest of blood in the branches of the pulmonary artery, which I have shown to be the essential cause of choleraic collapse."

For the present purpose, and for the sake of argument, we will grant the truth of the first proposition, that there is a definite cholera poison circulating in the blood. It remains to inquire as to the nature of the evidence in favour of the second, viz., that it is the special function of the cholera virus to contract the pulmonary artery. My own experience in cholera post-mortems, and much of the wider observations of others, tend to show that less blood than normal *is* sent to the lungs through the pulmonary artery, probably owing to a contracted state of that vessel. To this diminished supply of blood to the lungs, together with the general feebleness of the circulation, the *cyanosis* of the collapse stage is admitted to be mainly due; but it has yet to be proved that the contraction of the pulmonary artery is in any way out of proportion to that which occurs in the other arteries of the body. During life the already arterial pulse, or its absence, and the fact that when even large arteries have been cut down upon and opened they have been found contracted, and a flow of blood from them barely to be obtained, point to the condition of the systemic arteries; and according to my own experience in post-mortems on patients who have died from acute and rapidly fatal cholera collapse, the kidney and spleen are, for their parts, as anæmic as the lungs, though from the smaller proportion that the bloodvessels bear to the parenchyma in those organs

the diminution in actual weight is not so marked as in the case of the lungs. The liver does not reach quite its normal weight, but is fairly full of blood; which is readily explained by the small proportion that the blood supplied to it, through the muscular-coated contractile hepatic artery, bears to that which reaches it through the vena portæ,—less contractile, and bringing blood from acutely congested intestines. Were the collapse stage of cholera and consequent death due to the peculiar mode of asphyxia, resulting from the special contractile influence of the cholera poison on the pulmonary artery, although that presumed peculiar effect of the poison would account for the lungs themselves not being gorged with, but empty of blood, it would not explain the absence of the ordinary post-mortem appearances found after death from causes in which asphyxia took a prominent place, viz., engorgement with dark blood of all the internal viscera; yet this state, as far as my experience goes, is notably absent. On the other hand, I have found the kidneys and spleen, as well as lungs, appear to the eye anæmic, while the evidence of weight has shown them to be less full of blood than usual, so as not to obtain the average weight of those organs in health. In support of this statement I refer to the first six cases given in the body of this paper, especially to Case 4, and to the Tables I. and II. accompanying them. Leaving this question of the contracted state of the general arterial system, the point to look to is, does Dr Johnson, or, failing Dr Johnson, does any writer on cholera mention facts to prove that obstruction to the flow of blood through the lung, the consequent obstruction to aëration and the embarrassment to the breathing, invariably *precede* the other signs of the onset of cholera collapse, or, at least, take a marked lead among the symptoms? It is strange, that though the establishing of the fact of this precedence should be the very ground upon which Dr Johnson's theory ought to be securely based before it is taken as a starting point for dogmatic assertion, that it proves the true cause of cholera collapse, Dr Johnson does not bring forward a single instance from his own experience to show that such a priority exists in the symptom referable to the obstructed flow of blood through the lungs; nor does he quote the experience of others to supplement his own reticence on the subject. I have failed to find any record of such experience, and I have not myself seen anything to indicate that the signs of pulmonary obstruction in any way take the lead among the symptoms of cholera collapse; on the contrary, I have known the respiration be quite easy, and noted it as such, while cramps, coldness, small rapid arterial pulse, and inelastic skin, indicated the advance of collapse. Dr Johnson's reticence on this point is the more remarkable, as the rest of his arguments to prove that pulmonary obstruction *causes* collapse only go to establish, what has for many years been an admitted fact, that defective aëration of the blood is always *associated* with the algide state.

Even his ingenious argument—based on the continuance, during collapse, of some secretion of milk, a fluid requiring but li

oxygen for its formation, in which respect it resembles the bile, an abundance of which is always found in the gall-bladder,—contains nothing in support of the view that a cholera poison in the blood nduces contraction of the pulmonary artery, and that the obstruction hus produced is the "proved essential cause of cholera collapse." Such a cause has yet to be determined. I might, perhaps, better say causes; which would more nearly express what I believe to be he real state of the case, though I shall endeavour to prove that one deserves to be considered the main and leading factor. In earching for such a cause, several points must be kept in view,—irst and foremost, the cause must either be *proved* to be the ante-edent of the collapse stage, or, if not proved rigorously, the facts f the disease must render the presumption almost a certainty;—ext, the main cause, in conjunction with the secondary causes which calls into operation, must be sufficient to produce the symptoms of ollapse, in accordance with the known laws of the physiology of ne human body; and further, should the patient recover from the lgide state and do well, there must be presumptive evidence that ne cause had remitted,—and in such cases that die from secondary ffections, the evidence of post-mortem examinations should go to how that the cause of collapse had ceased to operate about the time nat the symptoms of collapse had passed off. The statement of hat I consider is the efficient cause of cholera collapse, and which believe fulfils the above requirements, I will defer till I have iven a brief abstract of the disease and detailed the post-mortem ppearances observed in six cases which died in profound and pidly fatal cholera collapse, and of four cases which recovered om the collapse and died from secondary affections. I would here ention that in relation to the post-mortem appearances, the rapidity the fatal result is of great importance. I believe that some of e discrepancies found in the descriptions of the morbid appear-nces met with after death in the collapse state would be explained y reference to the duration of that state prior to death. I regret at I cannot bring definite proof to support this opinion. If by the ght of the following cases one finds a definite and constant lesion esent in death during collapse—which state there is evidence of preceding—while it is less defined or even absent in those who e in a subsequent phase of the disease,—I think we shall be arranted, other things supporting the view, in considering such sion the cause, or intimately associated with the cause, of collapse.

Case 1.—Mary W., aged 45, stated to have had no premonitory diarrhœa; was ken with violent diarrhœa and vomiting at 6 A.M, 28th July, which con-ued all the morning, accompanied with severe muscular cramps. Admitted o cholera ward of the London Fever Hospital at 2.30 P.M. the same day. gide state was then marked; temperature in axilla 94⅘°; eyes sunken; lips l nails blue; skin inelastic; voice almost gone; pulse 108, extremely small l feeble; rice-water purging; vomiting frequent. Subsequently the collapse came more profound. Some of the stools passed were pink, and others ocolate-coloured from admixture of small quantities of blood; urine was npletely suppressed. Pulse at wrists and brachials ceased; respirations

became sighing, full, and slow; temperature at axilla fell to 94°; and woman died, without any sign of rallying, at 5 A.M., 29th July, having been ill for twenty-three hours.

Post-mortem Examination, same day, 10 A.M. *Heart*, right side moderately distended with black blood, which coagulated after exposure to air. *Lungs*, do not quite reach sides of thorax, to which they are bound by old stringy adhesions; tissue dry, and containing less blood than normal anteriorly, some congestion posteriorly, of hypostatic character; weights, right, 12¼ oz.; left, 11¼ oz. *Liver*, fairly full of blood, which was less fluid than ordinary; tissue, normal; gall-bladder contains 3½ oz. of bile; weight, 44 oz. *Spleen*, small, of pale colour, anæmic; weight, 3½ oz. *Kidneys*, healthy, contain rather less blood than normal; cortex, pale; capsule separates smoothly; stellate veins only slightly marked; weights, right, 3½ oz.; left, 3¾ oz. No urine in bladder, which was healthy. *Stomach*, mucous membrane only slightly injected; of pale pink colour. *Small intestines*, peritoneal surface minutely injected; pink; sticky from scanty amount of viscid secretion, only sufficient in quantity to give a slimy feel to surface and adhere in fine threads to finger; no definite lymph, but peritoneum presents a finely granular appearance from fulness of injected vessels; the coats of the intestines themselves are thickened, heavy, and deeply congested throughout; tint of congestion varies from moderate pink colour to bright red and venous purple colour, with patches of distinct ecchymosis. These conditions, though more or less present throughout whole length of small intestines, are most marked at lower end—one-third of ileum—where the solitary glands are hard, elevated, and congested, and some of Peyer's patches much injected and ecchymosed. Contents of duodenum and upper portions of jejunum: slimy mucus mixed with a very small amount of bile. Jejunum contained colourless and semi-opaque rice fluid, while the lower part of ileum was filled with a pink opaque fluid of creamy consistence, as though some blood had been mixed up with thin boiled ground-rice. *Large intestine*, several patches of dark venous congestion about transverse colon; but no uniform injection was present, and coats had not the swollen, sodden character of those of the small intestines; contents, fluid, chocolate coloured.

CASE 2.—Elizabeth W., aged 19, daughter of above, seized with violent purging and vomiting at 7 P.M., 27th July; cramps supervened; purging and vomiting continued frequent and free; was admitted, with marked symptoms of collapse, at 3 P.M., 28th July; temperature in axilla was then 94⅗°. After admission, girl frequently vomited small quantities, and had several watery, pinkish stools, as of thin mucus mixed with blood; urine was completely suppressed, and girl died in profound collapse, mind remaining clear, and pupils dilated till the last, at 9 A.M., 29th July. Duration of disease, thirty-eight hours. Temperature in axilla rose to 96° an hour before death.

Post-mortem Examination, 11 A.M., same day.—*Heart*, right side very fully distended with dark fluid blood, which soon and firmly coagulated; left cavities contained very small quantity of similar blood; tissue and valves healthy. *Lungs*, not at all collapsed, very buoyant, finely emphysematous; tissue, dry on section; froth very scanty and white; pulmonary artery apparently of normal calibre; weight, right, 9½ oz.; left, 8½ oz. *Liver*, normal; weight 45 oz. *Spleen*, not at all congested; weight 3½ oz.; section of rather paler colour than usual. *Gall-bladder*, full; contains 1½ oz. of viscid, dark bile. *Kidneys*, pale and anæmic; less blood than usual exudes on section; texture healthy; weight of each, 4½ oz. *Stomach*, contains three-quarters of a pint of greenish opaque fluid; mucous membrane pale and corrugated, as also is that of the first six inches of duodenum. *Small intestines*, below that point intensely congested, heavy, sodden, and swollen; injection varies in tint from pink, through bright red, to deeper venous colour; general appearance being almost exactly similar to those found in preceding case; most of the mucous membrane is coated with opaque mucus, somewhat granular at places, and mixed with blood where the injection is most marked; the peritoneal coat is tense, pink, and finely injected, giving the surface a finely granular look; no lymph, but surface too dry. Contents of small intestine varies according to region

somewhat as in Case 1. *Large intestine*, also much congested at places, but less uniformly; urinary bladder quite empty.

Case 3.—Martha D., aged 33, seized at about 12.30 A.M., the early morning of 3d August, with purging, vomiting, and painful cramps in stomach, which soon affected other parts. Profound collapse was established by 10 A.M., from which the woman never rallied. Mind was clear and pupils large; all the symptoms of the algide state were typically marked. Death took place at 11.30 P.M. same day. Six ounces of warm weak saline solution were injected into vein of right arm, several hours before death, without producing any marked effect beyond some præcordial distress, which soon passed off. Woman died more slowly than had been anticipated; duration twenty-three hours. History of state of bowels previous to attack was uncertain.

Post-mortem appearances.—Lungs, partly collapsed; some old adhesions and obsolete tubercle at upper fifth of both lungs; rest of lungs normal, except that section is too dry; weights, right, 13¾ oz.; left, 14 oz. *Spleen*, small; texture normal; weight, 4 oz. *Liver*, weight, 52 oz.; normal. *Kidneys*, anæmic to eye; section pale; otherwise healthy; weight, 4¾ oz. each. *Stomach*, contains some greenish fluid, and mucous membrane shows some patches of congestion. *Small intestines*, whole extent intensely and almost uniformly congested, heavy, and tumid; injection most marked on mucous surface, but peritoneum also affected; solitary glands are as large as mustard seeds; the intestines contain much flaky mucus coating the surface, and abundant rice-water fluid. Very slight partial congestion of large intestine. *Urinary bladder*, empty. *Gall-bladder*, full of black bile. *Uterus* contains fœtus of from five to six months.

Case 4.—Henry E., aged 60, was admitted, in moderately developed algide state of cholera, at 4 A.M., August 6. He then had cramps; was cold; lips and nails were blue; and skin was inelastic. From this state the old man rallied and went on well, without a bad symptom, till the night of August 11. On that day, a note taken of his state was to the following effect:—"Has had two natural-formed stools in the last twenty-four hours; tongue, moist, clean; pulse, —; man has good appetite and sleeps well." He was allowed a mutton diet, but without potato. His urine was passed freely, and had previously been found free from albumen. Unfortunately, the favourable course of this patient was suddenly interrupted. A scrubber of the ward listened to his request for further food than was allowed, and she, unknown to the nurse, gave him two good-sized cold and hard potatoes, which the man ate at 8 P.M. of the said 11th August. Between 10 and 11 P.M. the same evening, the man became very uneasy, was violently sick and purged. Collapse of profound and most characteristic choleraic character set in; pupils became dilated. Pulse ceased; cyanosis was developed; skin was again inelastic; and man died at 6.30 A.M. the next morning, August 12, about eight hours from the onset of symptoms, and ten and a-half after what was in all probability a fatal dose of cold potato. At the time of his death, the disastrous act of the scrubber was not known, nor was it confessed till the following post-mortem led to further inquiries being made.

Post-mortem.—Lungs, both decidedly collapsed, so as not nearly to fill the chest; all parts markedly buoyant; colour, dark from pigment; section dry and anæmic; weights, right, 9½ oz.; left, 10 oz. only. *Liver*, rather small, otherwise healthy; weight, 38½ oz., not cirrhotic or too fatty; contained fair amount of blood. *Spleen*, very small and anæmic; weight, 2¾ oz. *Kidneys*, anæmic; pale on section; contain less blood than normal; tissue healthy; weight, 3½ oz. each kidney. *Urinary bladder*, about quarter full of clear urine. *Gall-bladder*, half full of pale bile. *Stomach*, strongly contracted in centre; cardiac end filled with about 4 oz. of hard undigested potato, in lumps of size varying from a bon-nut to a small walnut; mucous membrane congested, of pink colour. *Intestines*, duodenum and upper part of jejunum were filled with fluid of thin gruelly character; the lower part of small intestines contained clear rice-water material; in the cœcum and large intestines some more undigested potato was mixed with a similar fluid; small intestines were congested nearly uniformly through almost their whole length; all the coats were swollen, heavy,

and turgid. Although the peritoneum was much and finely injected, the acute congestion of the mucous membrane was most marked, and to nearly its whole surface, much opaque glairy mucus adhered; solitary glands were hard and elevated; some of Peyer's patches were inflamed more and some less than surrounding parts; there was no trace of biliary tint in the intestinal contents.

Case 5.—Johanna K., ætat. 7. Father lies dead of cholera. Child was seized suddenly without any decided premonitory symptoms, at 4 A.M., August 8, with violent purging and vomiting, followed by cramps and other advanced symptoms of cholera. Was admitted into hospital at 2 P.M. same day, in a state of profound pulseless collapse, evidently moribund. Death took place at 4.30 P.M. Duration of severe symptoms, $12\frac{1}{2}$ hours.

Post-mortem.—Heart, right cavities full of soft dark clot. *Lungs*, weights, right, 6 oz.; left, 5 oz.; no collapse; rather less full of blood than normal, but condition of lung generally is healthy. *Liver*, normal; weight, 15 oz. *Spleen*, healthy; weight, 3 oz. *Kidneys*, anæmic; the *two* weigh $3\frac{3}{4}$ oz.; tissue healthy. *Stomach*, mucous membrane of pale pink colour. *Small intestines*, much injected throughout whole length; coats fleshy and heavy from active congestion; many of Peyer's patches deeply congested and ecchymosed on mucous surface, not extending through to peritoneal surface; solitary glands are large and prominent; much rice-water and gruelly fluid occupied the intestines, which were extensively coated with soft opaque mucus; not a trace of biliary tint in contents of intestines, though the *gall-bladder* was full of dark bile and the biliary ducts readily pervious. *Urinary bladder*, empty; urine was suppressed during life.

Case 6.—Anna H., ætat. $5\frac{1}{2}$, sent to cholera ward from a house where there had been several fatal cases of cholera. Child was dead on admission, though the distance to the hospital was short, and the ambulance a good one. Corpse had every feature of death having resulted from cholera collapse.

Post-mortem.—Lungs, buoyant; finely emphysematous; section dry; froth scanty and white; weights, right, $2\frac{3}{4}$ oz.; left, $2\frac{1}{4}$ oz. *Liver*, normal; weight, $11\frac{3}{4}$ oz. *Gall-bladder*, full of black bile. *Kidneys*, stellate veins or surface fairly full of blood and distinct; no minute congestion of secreting structure, which on section was rather paler than normal; weight of two kidneys, $3\frac{1}{2}$ oz.; no urine in bladder; spleen, normal. *Stomach*, normal, empty. *Intestines*, all coats intensely congested; injection of peritoneum very marked, and its surface rendered sticky by a slimy viscid secretion; mucous membrane of whole small intestine greatly congested, pink and ecchymosed; most of Peyer's patches are more affected than surrounding mucous membrane; intestines contain much rice-water material and opaque white mucus; perfectly free from biliary colouring.

Table I.—*Showing the Weights of some of the Viscera of four of the above adult cases, as compared with the normal weights, deduced from Dr Reid's and Mr Hutchinson's Observations.*

	Mary W. Case 1.	Eliz. W. Case 2.	Martha D. Case 3.	Henry E. Case 4.	Normal.	
					Male.	Female.
Lungs, right,	$12\frac{1}{4}$ oz.	$9\frac{1}{2}$ oz.	$13\frac{3}{4}$ oz.[1]	$9\frac{1}{2}$ oz.	24 to 21 oz.	17 to 15 oz.
„ left,	$11\frac{1}{4}$ „	$8\frac{1}{2}$ „	14 „	10 „		
Kidneys, right,	$3\frac{1}{2}$ „	$4\frac{1}{2}$ „	$4\frac{3}{4}$ „	$3\frac{1}{2}$ „	$4\frac{1}{2}$ to 6 oz.	4 to $5\frac{1}{4}$ oz.
„ left,	$3\frac{3}{4}$ „	$4\frac{1}{2}$ „	$4\frac{3}{4}$ „	$3\frac{1}{2}$ „		
Spleen,	$3\frac{1}{2}$ „	$3\frac{1}{2}$ „	4 „	$2\frac{1}{2}$ „	5 to 7 oz.	4 to 10 oz.
Liver,	44 „	43 „	52 „	$38\frac{1}{2}$ „	48 to 58 oz.	40 to 50 oz.

[1] Old tubercle in both lungs—pregnancy.

Case 7.—Ann S., aged 32, admitted with cholera, 4 P.M., 1st August. Severer symptoms had lasted twelve hours; marked collapse on admission; pulse 130, barely to be felt; algide symptoms marked; temperature in axilla 94°; marked cyanosis; pupils dilated; some of liquid stools passed had pink, bloody tint. From this state, woman rallied, and gradually improved; temperature rose to normal height in axilla. *4th August*, pulse was 86; skin was natural, and warm; woman had good appetite, and expressed herself as feeling well; passed two green pultaceous stools. Subsequently this patient went back; she passed into a low state, with dry tongue; made a good deal of phosphatic urine, free from albumen; a rash of a mixed, rubeoloid, and urticaria character came out on arm and trunk; muttering delirium supervened, associated with signs of pneumonia. Death occurred 11th August, on the eleventh day of her illness.

Post-mortem.—Heart, pale fibrinous clots, interlaced, on both sides of heart, extending to pulmonary artery and aorta, and forming casts of valves. *Lungs*, right, all but quite the apex and anterior margin, solidified by coalescence of lobular pneumonia, some of lobules softening in centre; weight, 35 oz. Left, several small patches of lobular pneumonia, and some hypostatic congestion posteriorly; weight, 18 oz. *Liver*, nutmeg condition very pronounced at places, with much general fatty degeneration; weight, 61 oz. *Spleen*, normal; weight, 5 oz. *Gall-bladder*, full of pale, clear bile, intermingled with opaque, turbid, glairy mucus; lining membrane is markedly congested, granular, and has small flakes of lymph on it. *Kidneys*, pale, and flabby; cortex, full, and opaque; bulges on section; but neither cut surface, nor that from which capsule readily separates, is granular; weight of each, 6¼ oz. *Stomach*, some dull, pink injection at cardial end. (A considerable amount of stimulant was given for thirty-six hours prior to death.) *Intestines*, marked absence of the state of general congestion and turgidity of the coats noted in the former cases, but several patches of local congestion were present, of dark venous colour, at various points of duodenum, jejunum, and ileum. At these spots the mucous membrane was swollen and soft, but not ulcerated, and pultaceous green fæcal matter adhered to them. At two or three points of lower end of ileum, which was healthy, the intestine was contracted upon small portions of formed yellow fæces. One broad patch of purple congestion was present, in transverse colon. Elsewhere, large intestine had healthy appearance. There were yellow fluid fæces in sigmoid flexure and rectum.

Case 8.—Martha S., aged 4½, daughter of above, was admitted same day; suffering from violent rice-water purging and vomiting; eyes were sunken; surface cold; lips and nails blue; child had painful cramps; pulse was small, rapid, and feeble, but could be counted at all times. From this state, child rallied, and regained normal temperature; lost blueness, and rice-water character of stools; but, in spite of treatment, chronic diarrhœa, of fæcal nature, came on, which exhausted the child, who died on the eighth day of her illness.

Post-mortem.—Heart, pale, firm fibrinous clots found on both sides of heart, and extending up pulmonary artery and aorta, forming casts of the valves. *Lungs*, scattered points of lobular pneumonia, of very trifling amount, in both lungs; no massive consolidation; no tubercle; *bronchial glands*, full of yellow tubercle; weights, right lung, 5 oz.; left, 4 oz. *Liver*, normal; weight, 41½ oz. *Kidneys*, pale, but cortex full, and stellate veins strongly marked; weight of each, 2¼ oz. *Spleen*, normal. *Urinary bladder* contained 3 or 4 oz. clear urine. *Gall-bladder*, full of pale bile, mixed with glairy, opaque mucus, adhering to congested lining membrane. *Stomach*, normal. *Intestines*, nowhere at all congested; coats, thin, pale, and translucent; scant amount of green, slimy, pultaceous matter in small intestines; yellow fæcal liquid in large.

Case 9.—George B., aged 26, was attacked with cholera, 29th July; vomiting and purging was free. He rallied from collapse stage, but colourless purging continued to end of fourth day; great prostration remained; low delirium set in, at times noisy; urine, containing a trace of albumen, was freely secreted, but retained; left pupil was somewhat dilated; respirations became slow and noisy; low pneumonia came on, and man gradually sank, without

having any further diarrhœa, at 2 P.M., 23d August, on the seventh day of his disease.

Post-mortem.—Head, much sub-arachnoid fluid; no opacity of membrane, considerable effusion of clear fluid in both lateral ventricles; brain substances of somewhat too pink a tint; no deposit, lymph, or tubercle, anywhere, or any defined softening. *Lungs*, right, considerable lobular pneumonia at posterior base; portions, of size of nut, coalescing at various points, not softening at centre; weight, 30¼ oz.; left, *lobular pneumonia*, of less amount, present; smaller masses, and more scattered; weight, 25 oz. *Heart*, firm decolorized clots pass up from both sides of heart in pulmonary artery and aorta, and form casts of valves; valves, etc., healthy. *Liver*, normal; weight, 60 oz. *Gall-bladder*, full of black bile. *Kidneys*, of pale colour; cortex full; convex on section, and too opaque; capsule separates smoothly; no evidence of old disease; weights, right, 6¾ oz.; left, 6 oz. *Stomach*, patches of dull pink injection. *Intestines*, a few patches of congestion noticed on mucous surface, some foot or so above cœcal valve, but not strongly marked; notable absence of the turgid, swollen condition of the coats of the small intestines; duodenum and upper part of jejunum found half-filled with firmish, pultaceous, slimy, green matter; lower end of ileum empty. *Large intestine*, large patches of dark, purple congestion, with swollen condition of mucous membrane, and slight enlargement of solitary glands observed in ascending and transverse colon.

CASE 10.—Mary H., æt. 45, a confirmed drunkard, after one day of moderate diarrhœa, was attacked at 9 P.M., 8th August, with vomiting and more violent purging; severe cramps supervened; collapse set in the next day at about noon, and woman was admitted into hospital at 6 P.M. the same evening, with well-marked symptoms of cholera collapse. Woman rallied very favourably in the succeeding thirty-six hours. A note, taken on 11th August, 10 A.M., states :—" Occasionally vomits a little green matter; keeps down a fair amount of nourishment; voice has returned; pulse, 76, distinct, and of fair power; temperature in axilla, 96°; feet warm; no blueness, or aspect of collapse; tongue moist, and furred; is stated to pass a fair amount of urine, with several fæcal motions; dozes off and on; the pupil of only eye small. Through the succeeding day and night patient got worse again, but there was no return of collapse; pulse remained at 80, feeble; respirations became slow, and sighing; there was restless, active delirium, alternating with snatches of sleep; constant fæcal diarrhœa, and rapid failing of powers; no urine was noticed to have passed. Death took place 8.30 A.M., 12th August.

Post-mortem.—Lungs, old pleuritic adhesions, and some obsolete tubercle, with firm cicatrices at both apices; no collapse; right, lower lobe partly becoming solid from coalescence of points of lobular pneumonia; weight, 22½ oz.; left, less of scattered lobular pneumonia at posterior base; tissue friable; weight, 16 oz. *Heart*, visceral and parietal pericardium very loosely glued together over base of heart and large vessel; lymph very soft and aplastic, shading off into a glutinous semi-purulent fluid, of which about ʒvi. were present; large, firm, and pale clots form casts of pulmonary and aortic valves, and occupy cavities of heart. *Liver*, of normal appearance; weight, 46 oz. *Spleen*, healthy. *Kidneys*, fairly full of blood, but not at all deeply congested; capsule tears tissue on being removed; cortex too opaque at places, and bulges on section; other points are firm, and contracted; a few minute cysts on surface; weights, right, 4½ oz.; left, 5½ oz. *Stomach*, some injection, of pink colour, at cardiac end, but slightly marked; contains green, grumous fluid. *Intestines*, from the outside, the small intestines do not appear to be congested; they are not thick, and fleshy to feel; nor on the mucous surface is the congestion particularly marked, except at two or three points of the ileum; some of Peyer's patches are moderately injected, but most are normal; solitary glands too prominent; the most congested part of canal is the upper, 4 inches below ileo-cœcal valves; parts below healthy; the upper part of small intestines contained much slimy bile-coloured mucoid matter; below, fæces were thin, and yellow. *Gall-bladder* contained dark bile; bladder half-full of clear urine.

TABLE II.—*Showing Weights of some of the Viscera of the Three Adult Cases who died during the Non-collapse Stage of the Disease, as compared with Normal Standard. This Table may also be compared with Table No. I.*

	Case 7. Ann S.	Case 9. George B.	Case 10. Mary H.	Normal.	
				Male.	Female.
Lungs,[1] right, .	35 oz.	30¼ oz.	22½ oz.	24 to 21 oz.	17 to 15 oz.
„ left, .	18 „	25 „	16 „		
Kidneys, right,	6¼ „	6¾ „	4½ „	4½ to 6 oz.	4 to 5½ oz.
„ left, .	6¼ „	6 „	5½ „ [2]		
Liver, . . .	61 „	60 „	46 „	48 to 58 oz.	40 to 50 oz.
Spleen, . . .	5 „	?	?	5 to 7 oz.	4 to 10 oz.

TABLE III.—*Comparison of the Weight of Organs of a Child, aged 4½, who died in Non-collapse Stage, with those two others, aged 5½ and 7, who died in Collapse.*

	Non-Collapse.	Collapse.	Collapse.
	Case 8. Martha S., æt. 4½.	Case 6. Anna H. æt. 5½.	Case 5. Johanna K., æt. 7.
Lungs, right, .	5 oz.	2¾ oz.	6 oz.
„ left, .	4 „	2¼ „	5 „
Kidneys, right,	2¼ „	1¾ „	1⅞ „
„ left, .	2¼ „	1¾ „	1⅞ „
Liver, . . .	14½ „	11¾ „	15 „

The study of the post-mortem appearances of the above cases, and the comparative tables appended to them, of the weight of the viscera in relation to the period at which death occurred, and the average healthy weights of the same organs, points unmistakably to a cause of collapse, fulfilling the conditions mentioned above. This cause is intense congestion and inflammation of the whole length of the small intestines, causing them to be fleshy, pink, and heavy, with sticky glutinous matter on the injected, finely granular peritoneal surface in some instances, and with general injection and œdema of the mucous membrane and patches of ecchymosis in *all* of those cases which died in the collapse stage of the disease. This condition is associated, in the same class of cases, with a more or less anæmic state of the lungs, kidneys, and spleen, while the liver maintains about its normal appearance,—a fact probably due to its main supply of blood being derived from the but little contractile vena portæ. During the existence of this condition of the internal organs, we know that before death the superficial arteries and cutaneous veins are contracted and empty of blood, and the skin is shrunken. If this state were produced by a special poison acting on the pulmonary artery, or upon the nerve centres even, which

[1] More or less pneumonia present in all.—*Vide* cases.
[2] Some old contractile disease present.

regulate the supply of blood in the arteries of the body generally, how does it happen that the nerves and arteries of the intestines are either exempted from the peculiar influence, or are acted upon in just the reverse manner; the small intestines (and in a less degree the large intestines) being the only portion of the body to which an active determination of blood has occurred? In those cases in which death had taken place after the collapse stage had passed off, although other morbid appearances were found, the condition of the intestines was markedly different; only patches of local congestion were present, as though some portions had remained passively congested, while the diffuse bright injection and turgid swollen condition had entirely or almost completely disappeared.

The chain of causation appears to be the following:—A poison in the alimentary canal acts there as a direct irritant, causing more or less rapidly-developed congestion and inflammation of the whole small intestine, to which much blood is determined. The intestine, meaning by the term the tissue of the various coats, becomes full and turgid, and acutely œdematous, whereby a strong rapidly-developed impression, resulting in shock, is made upon the innumerable and widely-spread branches of the sympathetic from the solar plexus, by which the duodenum, jejunum, and ileum are supplied. The well-known intimate connexion of the solar plexus with the splanchnic and pneumogastric nerves, and also with the posterior roots of the corresponding spinal nerves, insures the diffuse spread of this impression, amounting to a shock, from which results a general slow contraction of the organic muscular fibres of the whole arterial system, affecting not only the pulmonary artery, but the systemic arteries, including those of the kidneys and spleen, which are found anæmic after death, and also including in all probability the hepatic artery, though from the peculiar nature of the circulation in the liver the effects there are less manifest.[1] Before this view can be received, there are several propositions to which we must on good grounds assent.

I. *That there is an inflammation of the intestines.*—We are so accustomed to associate the presence of inflammation with a hot febrile state of skin that the cold stage of cholera is likely to throw us off our guard. The algide state so rapidly supervenes in most cases that scarce any come under proper observations in hospitals soon enough to admit of the temperature in the axilla being taken. In but one case have I had an opportunity of forming an opinion as to whether the temperature of the skin was raised at quite the commencement of cholera, and I regret that I did not then bring the matter to the test of a thermometer. It was that of a night nurse on duty in the cholera ward of the London Fever Hospital. She was seized at 2 A.M. with violent purging and vomiting. Coming myself direct from a warm bed at 3 A.M., the temperature

[1] The ductus communis cholidochus, in all probability, is included within this contractile influence, and thereby is explained the absence of bile from the intestines during the continuance of collapse.

of her skin was felt by me to be decidedly above the normal. I feel confidence in asserting this, as I have had much experience in taking body temperatures, and have been accustomed to verify judgments based on sensation by appeal to the thermometer. With the above exception, evidence of the temperature of the skin at the earliest stage of cholera is absent.

During the algide stage the phenomena of the disease, the scant supply of blood to the skin through the arteries, etc., partly account for the coldness of the surface; but, just as by the use of the thermometer, Dr Ringer established that the temperature of the blood was considerably raised even during the cold stage of the ague, so other observers, by taking the temperature within the vagina or rectum of patients in the collapse of cholera, have been able to bring abundant proof that even at that period, while the heat of the axilla only marks 95°, in those orifices it mounts to 101° to 103° F.

To illustrate this point I give in a tabular form the results of several thermometric observations made by Mr Charles E. Squarey, of the London Fever Hospital, simultaneously in the axilla and rectum or vagina of two patients, husband and wife, who died from the effects of progressive choleraic collapse. They corroborate observations made in the course of the epidemic of 1848.

Temperature in axilla, ...98°	96$\frac{2}{5}$°	96$\frac{4}{5}$°	96$\frac{1}{5}$°	98°	96$\frac{2}{5}$°
Do. vagina, ...101$\frac{4}{5}$	103$\frac{4}{5}$	102$\frac{4}{5}$	101$\frac{4}{5}$	101$\frac{2}{5}$	100$\frac{3}{5}$
Temperature in axilla, ...97°	97$\frac{3}{5}$°	96$\frac{4}{5}$°	98$\frac{4}{5}$°	98$\frac{1}{5}$°	
Do rectum, ...100	102$\frac{2}{5}$	103$\frac{1}{5}$	101$\frac{1}{5}$	100	

In all probability, the elevation of temperature of the surface of the body, found after death in some cholera corpses, may be accounted for by the conduction outwards of the heat of the interior, aided by the passage of hot blood from the internal large arteries through the relaxed small arteries and capillaries of the surface after death. This may also account for the muscular movements of some cholera corpses.

The following observations, made by Mr Squarey and myself on the body of a man aged sixty-seven, who died in cholera collapse, show that if at the time of death there is no marked difference between the internal and external temperatures, the elevation of temperature of skin is very trifling after death:—

Time.	Axilla.	Rectum.	Remarks.
3 P.M.	96°	98$\frac{2}{5}$°	
3.20	95$\frac{2}{5}$	98$\frac{1}{5}$	Time of death.
3.30	96	97$\frac{4}{5}$	Muscular movements observed of moderate extent. Hand moved towards face.
3.40	95$\frac{4}{5}$	97$\frac{3}{5}$	
3.50	95$\frac{4}{5}$	97$\frac{3}{5}$	
4	95$\frac{3}{5}$	97$\frac{2}{5}$	
4.10	95$\frac{2}{5}$	97	
4.20	95	96$\frac{4}{5}$	
4.30	95	96$\frac{3}{5}$	
	Interior of abdomen.		
4.40	95	98	
4.50	94$\frac{3}{5}$	98	
6		96$\frac{2}{5}$	After removal of body to dead house.

Further observations are required on this point.

The evidences of inflammation of the intestines, derived from post-mortem appearances, are very strong. The swollen condition, the intense hyperæmia and inflammatory œdema, resulting in some points in ecchymosis; the tenacious and granular mucus, sometimes tinged with blood, which coats the mucous membrane; the presence of false membrane and ulceration, which have occasionally been recorded; the admixture of altered epithelium and exudation cells; all these conditions found after death in the collapse stage, most surely show that the intestine was acutely inflamed during life. If they do not indicate this, we must abandon conclusions based on similar evidence concerning other diseases admittedly inflammatory.

The character of the rice-water stools is also markedly in accordance with what is known to be the peculiar qualities of inflammatory exudations, containing, as they do, a large proportion of chlorides of sodium and phosphates. Mr Simon, in his article on "Inflammation," in the "System of Surgery," p. 27, vol. i., says—"The great characteristic of inflammatory effusions is their excess of chloride of sodium and of phosphates." The well-known disappearance of the chlorides from the urine during the exudation stage of pneumonia indicates that their abundant effusion is, if not peculiar to, yet distinctly connected with the exudation from inflamed tissues. I have frequently had opportunities of testing the thin discharges from the visibly inflamed nasal mucous membrane of scarlatina patients, and have always found the fluid loaded with chlorides. From the above considerations I consider we are warranted in assenting to the proposition, that there *is* an inflammation of the intestines.

II. *That symptoms referable to this inflamed state of the intestine precede those associated with collapse*,—is the next proposition to which we must agree.

The testimony of writers on cholera is unanimous on this point. A peculiar sinking feeling—at first not amounting to a pain, yet very distressing, referred to the epigastrium—followed by painful cramps of the intestines, violent sickness and purging, the products of inflammation being found in the stools, are in the vast majority of instances present for a time, varying from one to several hours prior to the onset of collapse symptoms. The above group of symptoms clearly point to the intestines being the seat of the disease, and taken in connexion with the post-mortem appearances, the conclusion that such is the case is irresistible. Even in those cases in which death has rapidly resulted from collapse, before any external rice-water purging has been established, it is clearly stated that the intestines have contained the peculiar product, and have themselves shown the morbid appearances which, for reasons given above, must be considered as resulting from inflammation. There is no case on record that I can find, however rapidly fatal, in which conclusive evidence of an affection of the intestines, in accordance with the descriptions given above, was not present whenever looked for. Intimately allied, as an

evidence of causation, with this question of priority of the inflamed condition of the intestine to the onset of collapse, is that of the abatement of the symptoms referable to any acute inflammatory action in the bowels, as the collapse passes off, and when reaction is established. Although some sickness, diarrhœa, and unpleasant sensations, which have the intestines for their seat, remain, still the intensity of all these symptoms is then greatly mitigated, and their character altered. Moreover, even when death occurs, if it take place after, even shortly after, the collapse has passed off, from secondary affections, the post-mortem appearances indubitably show that the severity of the inflammation of the small intestines has gone by, as evidenced by the coats having returned to their normal thickness, the general injection and inflammatory œdema having passed off, a few patches of localized congestion of a darker character, and showing signs of less activity, alone remaining. In support of this statement, I would refer to the post-mortem examinations of Cases 7, 8, 9, and 10, which died in the non-collapse state, especially to Case 10, Mary H., who died very shortly after the reaction from collapse had been effected. It is not, however, entirely on the evidence of those few cases, that I rely, but I will quote a few lines of the resumé, in the College of Physicians' Report on Cholera, on the state of the small intestines after death in the stage of reaction. "The mucous membrane of the small intestine was generally pale or only slightly hyperæmic, and the pulpy œdematous condition of the tissue and intestinal glands, occurring in the algide stage, had much diminished or entirely disappeared." This quotation is important, in that by implication it confirms my experience of the post-mortem appearances found in those cases which died in the collapse stage.

III. *That the inflamed condition of the small intestines, and the morbid impression emanating from them, made upon the ganglionic and sympathetic system of nerves, is a* SUFFICIENT *cause of collapse, in accordance with the known laws of the physiology of the human body*,—is the remaining proposition to be considered.

To establish the great probability of the sufficiency of the cause, we must review as many as nearly as possible parallel cases as we can find, in which shock to the system, radiating from the alimentary canal and abdominal viscera, has induced collapse coinciding in many of the leading symptoms with that seen in cholera. I am well aware, before starting on the inquiry, that no exact parallel is to be obtained, for were it to be found it would be cholera. A general strong similarity is all that we require—all, in fact, that could advance the argument. Exactness of the parallel would leave us in consideration of a barren identity.

Although the nerves supplying the large intestine are not derived from the solar plexus, and have not such intimate connexions and sympathies with the great ganglionic nerve centres supplying the heart, lungs, and arterial systems, as those distributed to the small intestines, I am of opinion that the following quotation from the

"Treatise on the Practice of Medicine," by Dr George Wood,—in which, when treating of dysentery, he thus describes the symptoms associated with extensive inflammation of the large intestines,—shows that there is an instructive analogy between the symptoms of some severe cases of dysentery and of cholera. The quotation runs thus: "Occasionally, in cases of some severity, the vital forces sink temporarily under the violence of the impression made upon the nervous system. The patient experiences an indescribable painful feeling of hollowness or sinking in the abdomen, attended with cold damp skin, feeble and almost thread-like pulse, and sometimes nausea and vomiting. Sometimes, however, from the extent and severity of the inflammation, symptoms of depression appear at the outset, and the system never fairly reacts. Here the same condition appears to exist continuously which has been above described as occurring occasionally in milder cases. The nervous system yields to the violence of the disease. The patient has throughout a very small and frequent feeble pulse, a pale, cool, and clammy skin, anxious and sunken features, and a somewhat livid and purplish appearance under the eyes, about the lips, and at the roots of the nails; while, at the same time, there is extraordinary violence of the local symptoms—such cases prove fatal in a few days." It is, however, among the symptoms produced by the action of irritant poisons, whether mineral, animal, or vegetable, upon the alimentary canal, that one would expect to find the closest parallel. Inquiry in this direction will give us some remarkable analogies. Thus Dr Guy, in his "Forensic Medicine," p. 390–1, has the following remarks on the effects of arsenic: "When the poison proves rapidly fatal, death commonly takes place by collapse or coma. . . . . There is a cold and clammy skin, extreme prostration of strength, the pulse very frequent and almost imperceptible. . . . . The mind, as in most cases of poisoning by arsenic, is unimpaired, but there is some approach to coma, slight cramps and convulsions, and death without reaction. Sometimes this state of collapse is accompanied by constant vomiting, and profuse purging. In this class of cases, death often takes place in four or five hours, and it is rarely delayed beyond twenty hours." Farther on, after detailing the post-mortem appearances of inflammation found in the stomach in cases of poisoning by arsenic, Dr Guy remarks, "The inflammation generally extends to the duodenum and commencement of the other small intestines, and occasionally affects the whole length of the alimentary canal, being most conspicuous in the lower bowel." While considering this subject of the analogies of cholera, my attention was directed to the very interesting work on "Cholera in its Home," by Dr Macpherson,[1] in which several instances are so graphically given, that I cannot do better than quote them in the language of his book. "The following is an abstract of the symptoms of poisoning by tartar emetic.[2]—Copious

[1] Late Deputy-Inspector-General of Hospitals, H. M. Bengal Army.

[2] From Beck's Medical Jurisprudence, 1838.

vomitings; frequent hiccup; burning heat in the epigastric region; colic; copious stools; syncope; small accelerated pulse; cold skin, but sometimes intense heat; difficult breathing; vertigo; fainting; convulsive motions; very painful cramps in legs; prostration of strength; and death." Dr Sandbusch thus describes the symptoms resulting from eating putrid flesh:[1] "Violent thirst; nausea and vomiting; abdomen at times spasmodically tense, at others, soft; constipation, or oftener diarrhœa; strangury, almost amounting to retention; difficulty of swallowing, and of respiration; headache, or swimming of the head; blueness of the face; dilatation of the pupils; small weak pulse; numbness of points of fingers; coldness of extremities; and marked sinking of the vital powers. In case of recovery the collapse and characteristic symptoms often suddenly cease." I will quote one further instance from the same author:—"An example of a vegetable irritant is afforded in the effects produced by croton oil. At the end of three-quarters of an hour after the oil had been swallowed the skin was cold and covered with sweat, the pulse and action of the heart scarcely perceptible, respiration difficult, the points of the fingers and toes, the parts around the eyes and lips blue, as in malignant cholera; but no vomiting. In an hour and a-half there were excessive and involuntary alvine discharges, sensation of burning at œsophagus, skin colder, respiration and circulation difficult, the cyanosis extended over the whole body, the skin became insensible, and death occurred with symptoms of asphyxia four hours after the poison was swallowed. In the latter case of a vegetable purgative, the symptoms approached wonderfully close to those of cholera." I have not been able to meet with any description of the post-mortem appearances found after croton-oil poisoning. To determine this point to some extent, I gave a poisonous dose of the oil to a dog, which caused violent vomiting and scanty bloody stools, attended with considerable depression. Before giving the poison, the temperature in the rectum was 102°. Three hours afterwards the temperature had fallen to 98½°. The animal was *killed* twelve hours after the oil was given. The sole morbid lesions discoverable were intense congestion of the stomach, which was empty, and of dark port-wine tint. The small intestines were much less affected, but presented a pinker tint, and some of the Peyer's patches were more congested than those of a healthy dog with which they were compared. They contained only a small amount of colourless fluid. The large intestine presented just the appearance, throughout its whole length, as that mentioned as found in the stomach, and contained glairy bloody mucus of the same character as the stools prior to death.

In connexion with all the foregoing cases, the remark of Caspar,[2] in his Forensic Medicine, "that the phenomena attendant on many kinds of poisoning resemble those of Asiatic cholera," is worthy of

[1] Allegemeines Repertorium, 1846.

[2] Sydenham Society's Translation, by G. W. Balfour, M.D., vol. ii. p. 67.

attention; and, taking all the above cases into account, I am of opinion that, at least, a case of the greatest probability is made out of the *sufficiency* of the inflamed state of the intestines to account for the development of the algide symptoms of cholera, in accordance with the line of causation mentioned in a former paragraph.

I believe that the foregoing arguments establish, as nearly as the question admits of anything like proof, the chain of causation of cholera collapse; and I believe it to be in accord with all the phenomena of the disease, and the facts of pathology as far as yet known. There are two or three further points to be considered, which all tend to confirm the views I have stated. The view, held by some, that the phenomena of collapse and the contracted state of the arterial system are due to the continued action of a sui-generis poison, in the blood, upon the nervous centres, must be considered. This proposition is maintained by the strongest advocates of so-called eliminative treatment; yet it is a matter of the widest experience that, setting aside exceptional cases, profuse rice-water diarrhœa and frequent free vomiting have been followed by most profound collapse, just at the period when it would be presumed that some at least of the poison had been eliminated. In reply to this, it would be answered that the poison is a ferment which increases itself in the blood; and in those cases where collapse follows free purgation and vomiting, it results from the poison multiplying faster than the elimination gets rid of it. Now, this mode of behaviour of the cholera poison rests on pure assumption, and though mere assumptions are useful as a tentative method of explanation, they must give way if opposed to facts which support another explanation, itself based on the ascertained facts of the disease. In illustration of this point, I would draw attention to those instructive cases in which a distinct relapse of cholera collapse has been evidently brought on by definite and gross errors of diet, committed at a time when the alimentary canal must have been left in a weakened state, although all evident constitutional effects of the poison had passed off. Two cases of cholera relapse, and both of this kind, have come under my notice. One was that of a young woman who was admitted into St Bartholomew's Hospital, under the care of Dr Andrew, suffering from profound cholera collapse. From that state she rallied, and went on without a bad symptom for some days, when, on the day before that on which she was to have left the hospital, her friends (?) brought her some bad indigestible pastry, of which she ate largely. Collapse of considerable urgency, and as characteristic of cholera as her original algide state, supervened, from which she again rallied, after castor oil had expelled the offending matters, together with considerable amount of rice-water matter. The other case was that given at page 9, Case 4., Henry E., and is well worthy of attention. Till the unfortunate meal of two hard cold potatoes, the man was well in all his bodily functions, as far as they could be investigated. The rapid development of vomiting, diarrhœa (rice-water), and collapse; the post-mortem evidence of lumps of potato

in an inflamed cardiac end of stomach; the fact that portions of the same irritant had passed indigested through the whole length of the small intestines, which were themselves intensely congested, œdematous, pink, and heavy, and contained large quantities of rice-water material; all these facts point to the potato being the cause of renewed intestinal inflammation, from which resulted the development of the algide state, which proved so quickly fatal. That bad pastry and hard lumps of potato should irritate and excite inflammation in an intestine rendered liable to assume a peculiar form of affection, can be readily understood, while it can hardly be conceived that either one or the other should increase the amount of the cholera poison in the blood.

I have in this paper purposely abstained from giving any prominence to the treatment of the cases above recorded, because I have not pursued quite the same method with all, and no useful end could be served by stating the treatment, unless one could give a tabular statement of very large numbers. I would, though, make one or two observations on the use of the drugs castor oil and opium, which seem to bear closely on the conclusions at which I have arrived. If the sketch given of the pathology of cholera and the mode of causation of collapse be correct, it tends to establish that castor oil is contra-indicated, except as an aperient to remove undigested material from the alimentary canal at the commencement of the premonitory diarrhœa, or even later in the case, should the presumption of undigested matters in the intestines be strong; but then it should be borne in mind that the castor oil, irritant as it is—though a mild one—stands in the position of the lesser of two evils. After collapse has passed off, in certain cases castor oil is again indicated to remove the very peculiar slimy and pultaceous green matter which accumulates in the duodenum and jejunum, and must be supposed, from its abnormal character and consistence, to require removal. As to the use of opium, the beneficial influence of this drug, which has so many and diverse authorities in its favour, and which is recommended by the College of Physicians in all stages of cholera short of established collapse, may be explained in accordance with the views I have stated. It is *the* drug always given to protect the system from shock, or lessen its effects when it has been sustained. Opium circulating in the blood seems to induce a state of the nervous centres, the very opposite of what must be their condition in the collapse of cholera, if we may take the striking contrast, presented by the symptoms associated with the development of that state and those of advancing opium poisoning, as an indication for such a conclusion. This, I think, we may fairly do. It is, though, but little use theorizing on the subject of treatment. Such a process may, with advantage, suggest lines of treatment, but their success must be established by very wide and impartial collation of a large number of assorted cases.

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